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THE ROLE OF MULTIPLE V CHEST AND aV LIMB LEADS IN ROUTINE CLINICAL ELECTROCARDIOGRAPHY

The life span of electrocardiography includes several phases, of which the first was marked by its introduction into the clinic, and its recognition as the final arbiter in the diagnosis and clarification of cardiac arrhythmias. It soon became apparent that certain alterations in contour of P, QRS, and S-T-T configurations might provide valuable information in the diagnosis of heart disease.

The introduction of precordial leads two decades ago marked the second major phase. Although a lead from any extracardiac point such as the chest wall reflects in varying degree the potential variations of every part of the heart, the magnitude of influence of any given element varies inversely as the square of its distance from the electrode. Theoretical and practical experience have shown that the chest leads ordinarily record complexes closely resembling those of direct epicardial leads obtained at corresponding positions. Thus, any single precordial lead predominantly reflects the potential variations obtained over a relatively small area of underlying epicardial surface. The need for recording multiple chest leads is thus made clear. Experimental studies demonstrated that the central terminal remains more nearly at zero potential during the heart cycle than other known connections; thus the V lead is preferable to CR, CL, CF, or CB connections as an indifferent electrode for recording chest leads.

Over a period of years, theoretical and empirical studies led to the realization that certain specific diagnostic patterns could be observed in the limb and chest leads in the presence of myocardial infarction, "heart strain," acute pericarditis, and acute and chronic cor pulmonale. It was, however, realized that the standard limb leads have a major disadvantage in that each is a bipolar connection representing potential differences between two points on the body without necessarily revealing the nature and magnitude of the electrical events occurring separately at each of these points. V limb leads, within limits, have accomplished the latter and have been made clinically more useful by a modification which enlarges the complexes. It is important to realize that neither V chest nor aV limb leads are actually unipolar and that the aV limb leads should have a five or ten thousand ohms resistance in each connection of the indifferent electrode to minimize currents flowing through the skin. Otherwise unequal differences in the resistances between electrodes and the skin of each extremity may make the potential of the indifferent electrode fluctuate too widely.

Within limits, any standard limb lead represents the mathematical difference in the potentials recorded in two respective aV leads, e.g. lead I represents the difference between leads aVL and aVR. It has been shown that the potential variations recorded by an aV lead, like a V chest lead, are dominated by those obtained at the epicardial surface nearest or facing that extremity. Since the patterns recorded in leads aVL and aVF usually closely resemble those of various precordial areas, the aV limb leads have established the relationship between the standard limb and V chest leads. Perhaps of greater significance is the impetus given by the aV limb leads to the concept of electrocardiographic ("electrical") position, which may be said to mark a third phase in the history of electrocardiography. Although initially pointed out many years ago, the importance of this concept has only recently been widely realized. The "electrical" position may be considered in analogy with the anatomic position, although these may actually be different. Both may rotate on three axes; a) clockwise or counterclockwise rotation on a longitudinal axis causing the transition zone in serial precordial leads to be shifted to the left or right, b) vertical or horizontal rotation on an anteroposterior axis causing the electrical axis in the standard limb leads to be shifted to the right or left, and c) shift of the heart's apex in a forward or backward direction on a transverse axis. In a recent study we found that the correlation between anatomic and "electrical" positions on an anteroposterior axis was good in almost 80 percent of 200 cases. A poor correlation was

obtained in patients with markedly enlarged hearts where the fluoroscopic estimation of anatomic position was difficult.

The introduction of a fourth phase, that of spatial vectorcardiography is already at hand. By this means the three dimensional sweep of the P, QRS, and S-T-T elements would be recorded as loops. It would replace multiple leads, except for focal effects and arrhythmias, and thus simplify electrocardiography.

For the present it is important to define the number and the specific leads which should be recorded routinely for accurate interpretation. In order to determine this, we undertook an empirical study in which standard and aV limb leads, and precordial leads V1 to V7, V3R and V4R were recorded. In each instance a 6 lead record (leads 1, 2, 3, V2, V4, and V5) was interpreted and the cases in which the 6 leads failed to corroborate a more accurate diagnosis established by an independent interpretation of the complete 15 lead record were analyzed. It was found that the 6 lead electrocardiogram failed to establish an accurate interpretation in 15 percent of the records obtained from 515 random patients. The aV limb leads proved essential for accurate interpretation of 6.8 percent of the 515 records and the additional precordial leads were essential to accurate diagnosis in 8.2 percent.

On occasion, non-specific abnormalities were limited to leads aVL or aVF, and conversely, the latter proved important in demonstrating that certain peculiar limb lead combinations considered abnormal per se were normal variants due to peculiar "electrical" positions. Our experience and that of others has shown the importance of aVL in the diagnosis of lateral wall infarction and of aVF in posterior wall infarction and occasionally in its differentiation from acute cor pulmonale. Furthermore, these leads were particularly important in the interpretation of left ventricular strain in various "electrical" positions; the strain pattern occasionally being limited to leads aVL or aVF.

It was found that leads V3R and V4R closely resemble each other and can be used interchangeably. Lead V4R is preferred because of simpler electrode placement. Lead V4R and V1 were found superior, indeed often essential to the correct interpretation of right ventricular strain, particularly in less advanced strain patterns, and in the correct localization of intraventricular conduction defects involving the right bundle branch system. However, lead V4R proved essential in this regard only in children. Lead V3 has been occasionally of value in the diagnosis of recent or remote myocardial infarction limited to the anteroseptal region of the heart. Non-specific abnormalities were occasionally limited to leads V6 and V7, particularly in records with a significant shift of the transition zone to the left. With such a shift, diagnostic patterns of left ventricular strain also were not infrequently limited to V6 and V7. However, V7 can be omitted, being essential in less than 1 percent of records. Serial precordial leads were sometimes necessary to establish the normality of individual chest leads. It has been our experience that statistical mathematical criteria applied to individual leads are of less value in determining normality than is a normal progression of direction and amplitudes of QRS and S-T-T complexes in the serial precordial electrocardiogram.

It should be emphasized that the recording of aV limb leads and of multiple precordial leads no longer belongs to the investigative realm. A wide experience has found both to be essential for accurate clinical electrocardiography. On the basis of our recent experiences, we have adopted and are recommending the practice of routinely recording both standard and aV limb leads and precordial leads V1 to V6. In children under ten years, lead V3 is omitted and V4R is added. On occasion further exploration with other precordial positions may be found necessary. If practicality demands, lead aVR may be omitted; the minimum precordial lead combination which might be considered satisfactory would be the routine recording of leads V1, V2, V4, and V6.

Finally it should be emphasized that the electrocardiogram is a valuable laboratory tool, but subject to all the fallacies of such a device and to the shortcomings of the personnel involved. It should be utilized as such and with no attempt to lend it more mathematical precision than it deserves.

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FURTHER PARTICULARS MAY BE OBTAINED FROM THE FOLLOWING SELECTED REFERENCES

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3. Myers, G. B. and Klein, H. A. The Relation of Unipolar Limb Leads to Precordial and Esophageal Leads. Am. Heart J., 35, 727, 1948.
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7. Goldberger, E. A Simple, Indifferent Electrocardiographic Electrode of Zero Potential and a Technique of Obtaining Augmented Unipolar Extremity Leads. Am. Heart J., 23, 483, 1942.
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PROGRAM
of the
TWENTY-THIRD SCIENTIFIC SESSIONS
of the
AMERICAN HEART ASSOCIATION
June 22-24, 1950
Gold Room, Fairmont Hotel, San Francisco, California

FIRST SESSION
1:30 P.M., Thursday, June 22

Chairman: Hugh McCulloch, American Council on Rheumatic Fever.

1. STAPHYLOCOCCUS BACTERIAL ENDOCARDITIS: A REPORT OF 18 CASES WITH OBSERVATIONS ON BACTERIAL RESISTANCE.
   George C. Griffith, David C. Levinson, and Harold E. Pearson, Los Angeles, California.
2. PREVENTION OF RHEUMATIC FEVER BY PROMPT PENICILLIN THERAPY OF HEMOLYTIC STREPTOCOCCAL RESPIRATORY INFECTIONS.
   Benedict F. Massell, George P. Sturgis, Richard B. Streeper, Thomas N. Hall, Joseph D. Knobloch, and Pliny Norcross, Boston, Massachusetts.
3. MURAL THROMBOSIS AND ARTERIAL EMBOLISM IN MITRAL STENOSIS: A CLINICOPATHOLOGIC ANALYSIS OF 51 CASES.
   Robert A. Jordan, Charles H. Scheifley, and Jesse E. Edwards, Rochester, Minnesota.
4. ELECTROKYMOGRAPHY IN THE DIAGNOSIS OF CHRONIC CONSTRICTIVE PERICARDITIS.
   Victor A. McKusick, Baltimore, Maryland.
5. THE NATURE OF AURICULAR FLUTTER IN MAN.
   Myron Prinzmetal, Eliot Corday, Robert Oblath, Isidor C. Brill, and H. E. Kruger, Los Angeles, California.
6. **LEWIS A. CONNER MEMORIAL LECTURE**—CURRENT STATUS OF TREATMENT OF BACTERIAL ENDOCARDITIS.
   Arthur L. Bloomfield, Professor of Medicine, Stanford University Medical School, San Francisco, California.
7. THE EFFECTS OF CORTISONE AND ACTH ON PERIARTERITIS NODOSA AND CRANIAL ARTERITIS: PRELIMINARY REPORT.
   Richard M. Shick, Archie H. Baggenstoss, and Howard F. Polley, Rochester, Minnesota.
8. THE EFFECT OF ADRENOCORTICOTROPHIC HORMONE (ACTH) ON RHEUMATIC FEVER.
   Albert Dorfman, Delbert M. Bergenstal, Earl P. Benditt, and Frances E. Moses, Chicago, Illinois.
9. THE RESPONSE OF THE HYALURONIDASE INHIBITOR AND MUCOPROTEINS IN ACTH THERAPY OF RHEUMATIC STATES.
   Forrest H. Adams, Vincent C. Kelley, Paul F. Dwan, and David Glick, Minneapolis, Minnesota.
10. THE EFFECT OF ACTH AND CORTISONE ON RHEUMATIC FEVER.
    Joseph J. Bunim, Currier McEwen, Janet S. Baldwin, and Ann G. Kuttner, New York, New York.

PANEL DISCUSSION: RHEUMATIC FEVER.

SECOND SESSION
9:00 A.M., Friday, June 23

Chairman: Robert W. Wilkins, Section on Circulation.

11. THE EFFECTS OF BLEEDING ON SODIUM EXCRETION, CARDIAC OUTPUT, AND GLOMERULAR FILTRATION OF NORMAL SUBJECTS.
    Seymour Eisenberg, B. B. Oliver, Tom Lombardo, and William Viar, Dallas, Texas.
12. COMPARISON OF REACTIVE HYPEREMIA WITH OTHER VASODILATING PROCEDURES FOR DETERMINING THE FUNCTIONAL CAPACITY OF THE PERIPHERAL CIRCULATION.
    Travis Winsor, Los Angeles, California.
13. A NEW HUMORAL PRESSOR SUBSTANCE PRESENT IN ARTERIAL HYPERTENSION.
    Henry A. Schroeder and Norman S. Olsen, St. Louis. Missouri.
14. THE RELATIONSHIP BETWEEN THE NATURE OF CHOLESTEROL TRANSPORT AND THE DEVELOPMENT OF ATHEROSCLEROSIS.
    John W. Gofman, Thomas P. Lyon, Frank Lindgren, Hardin B. Jones, and Harold Elliott, Berkeley, California.
15. **GEORGE E. BROWN MEMORIAL LECTURE**—SCIENTIFIC RESEARCH IS PROGRESS.
    Edgar V. Allen, Mayo Clinic, Rochester, Minnesota.
16. OXYGEN TENSION AND BLOOD FLOW OF THE SKIN OF NORMAL AND ISCHEMIC HUMAN EXTREMITIES.
    Hugh Montgomery and Orville Horwitz, Philadelphia, Pennsylvania.
17. CONTROLLED OBSERVATIONS ON LOW-SODIUM DIETOTHERAPY OF ESSENTIAL HYPERTENSION.
    A. C. Corcoran, R. D. Taylor, and Irvine H. Page, Cleveland, Ohio.
18. A CLINICAL METHOD OF ESTIMATING THE BLOOD PRESSURE IN THE SUBPAPILLARY PLEXUS OF THE HUMAN EXTREMITY BY THE METHOD OF ELEVATION AND REACTIVE HYPEREMIA.
    Rutherford S. Gilfillan and Norman E. Freeman, San Francisco, California.
19. THE MEASUREMENT OF PERIPHERAL RESISTANCE IN MAN.
    Milton Mendlowitz, New York, New York.

PANEL DISCUSSION: HYPERTENSION.

THIRD SESSION

1:30 P.M., Friday, June 23

Chairman: H. M. Marvin, President, American Heart Association.

20. THE URINARY AND BILIARY EXCRETION OF DIGITOXIN IN THE NORMAL AND CARDIAC PATIENT.
    Meyer Friedman, René Bine, Jr., and Sanford O. Byers, San Francisco, California.
21. PHYSIOLOGICAL DEMONSTRATION OF THE BLOCKING ACTION OF ATROPINE AND THE ANTIHISTAMINIC DRUGS ON CORONARY ARTERY VASOCONSTRICTION.
    Durwood J. Smith and Joseph W. Coxe, Rochester, New York.
22. PROCAINE AMIDE IN THE TREATMENT OF CARDIAC ARRHYTHMIAS.
    Herbert J. Kayden, Lester C. Mark, J. Murray Steele, and Bernard B. Brodie, New York, New York.
23. COMPARISON OF CARDIO-RESPIRATORY PERFORMANCE IN PATIENTS WITH DISEASES OF THE HEART AND LUNGS, AND NORMAL SUBJECTS UNDER UNUSUAL STRESSES DURING EXERCISE.
    Robert A. Bruce, Frank W. Lovejoy, Jr., Paul N. G. Yu, Marion E. McDowell, and William S. McCann, Rochester, New York.
24. THE ACTION OF STROPHANTHUS K ON THE CORONARY CIRCULATION AND CARDIAC OXYGEN CONSUMPTION OF THE NORMAL AND FAILING HEART IN VIVO.
    R. J. Bing, F. Maraist, J. Jacobs, R. Heimbecker, I. R. Goldman, A. Draper, and J. F. Dammann, Jr., Baltimore, Maryland.
25. SPECIAL LECTURE: PROGRAMS OF THE PUBLIC HEALTH SERVICE IN THE ATTACK ON CARDIOVASCULAR DISEASES.
    Leonard A. Scheele, Surgeon General, United States Public Health Service, Washington, D. C.
26. VIBRATIONS OF LOW FREQUENCY OVER THE PRECORDIUM.
    Franklin D. Johnston, Ann Arbor, Michigan.
27. EXPERIMENTAL AND CLINICAL EXPERIENCES WITH THE NEW ANTICOAGULANTS—TROMEXAN AND PARITOL.
    Irving S. Wright, C. W. Sorenson and Grafton Burke, New York, New York.
28. EXPERIMENTAL STUDIES ON THERAPY OF ACUTE PULMONARY EDEMA.
    Aldo A. Luisada, Chicago, Illinois.
29. TREATMENT OF CONGESTIVE HEART FAILURE WITH 50 MGM. SODIUM DIET: METABOLIC AND CLINICAL STUDY.
    Lloyd T. Iseri, Albert J. Boyle and Gordon B. Myers, Detroit, Michigan.

PANEL DISCUSSION: DRUG THERAPY

5:30 P.M., Friday, June 23

Annual Business Meeting.

FOURTH SESSION

9:00 A.M., Saturday, June 24

Chairman: Louis E. Martin, Chairman, Program Committee.

30. THE ELECTROCARDIOGRAM IN CONGENITAL HEART DISEASE: A PRELIMINARY REPORT.
    Oglesby Paul, Chicago, Illinois, and Gordon S. Myers, Boston, Massachusetts.
31. THE BALLISTOCARDIOGRAM: NORMAL STANDARDS AND DETERMINANTS OF THE WAVES.
    Richard Gubner, Manuel Rodstein, and Harry E. Ungerleider, New York, New York.
32. BLOOD FLOW AND OXYGEN CONSUMPTION OF THE BRAIN IN COARCTATION OF THE AORTA.
    Charles W. Crumpton, John H. Meyer, Julian Johnson, William A. Jeffers and Joseph H. Hafkenschiel, Philadelphia, Pennsylvania.
33. PRESSURE CURVES FROM THE RIGHT AURICLE AND THE RIGHT VENTRICLE IN CONSTRICTIVE PERICARDITIS.
    A. Tybjaerg Hansen, P. Eskildsen, and H. Gotzsche, Copenhagen, Denmark.
34. SPECIAL LECTURE: IS THE AMERICAN HEART ASSOCIATION PREPARED TO BECOME A NATIONAL VOLUNTARY PUBLIC AGENCY?
    William P. Shepard, Third Vice-President, Health and Welfare Division, Metropolitan Life Insurance Company, San Francisco, California.
35. THE CLINICAL SYNDROME PRODUCED BY VALVULAR PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM AND PATENT FORAMEN OVALE.
    Mary Allen Engle, Helen B. Taussig, and Caroline Bruines, Baltimore, Maryland.
36. A METHOD FOR VISUALIZATION OF THE CORONARY ARTERIES AND DEMONSTRATION OF THE SITE OF CORONARY OCCLUSION.
    James Helmsworth, Johnson McGuire, Benjamin Felson, and Ralph C. Scott, Cincinnati, Ohio.
37. DIAGNOSIS OF POSTERIOR MYOCARDIAL INFARCTION WITH THE AID OF SEMIDIRECT LEADS FROM THE BACK AND ESOPHAGUS.
    Charles H. Sears and Gordon B. Myers, Detroit, Michigan.
38. HEART DISEASE CASE FINDING WITH 70 MILLIMETER PHOTOFLUOROGRAPHIC FILMS.
    David D. Rutstein, Charles R. Williamson and Felix E. Moore, Jr., Boston, Massachusetts.

PANEL DISCUSSION: AIDS TO CARDIAC DIAGNOSIS.

7:30 P.M., Saturday, June 24

Annual Dinner

Gold Room, Fairmont Hotel, San Francisco, California

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